Case 1. Tissue from a cat.

(There are three sections of liver here which are all about the same.)

MICROSCOPIC DESCRIPTION: Liver: Approximately 50 percent of the section (1pt), excepting the subcapsular parenchyma, is effaced by dense bands of mature collagen (1pt) ranging up to 800um populated by plump fibroblasts. These bands of collagen bridge (1pt) and markedly expand (1pt) portal areas (1pt), often whorling around bile ducts and separating nodular remnants of midzonal and centrilobular hepatocytes. Small numbers of lymphocytes and plasma cells (1pt) are scattered diffusely throughout the fibrous bands, concentrating is moderate numbers around bile ductules, and occasionally form tertiary lymphoid structures (1pt). There is diffuse and often severe biliary hyperplasia (ductular reaction OK) (1pt); small ductules often lack lumina and larger ducts are often tortuous and ectatic. In some sections, branches of the portal veins are numerous and congested, while in others, they are rarely seen. (1pt) There is nodular proliferation (1pt) of the remaining hepatic parenchyma with marked distortion or loss of sinusoidal architecture and without distinct central veins (1pt). Hepatocytes at the edge of nodules or at the interface with advancing fronts of fibrosis are often compressed or atrophic; in more central areas; hepatocytes contain one more variably sized lipid droplets (1pt) which often compress and peripheralize the nucleus. Many nodules contain individualized or small aggregates of macrophages which have a brownish-yellow granular pigment (lipogranulomas,) (1pt). The hepatic capsule is undulant, with marked dilation of subcapsular lymphatics and veins (1pt).

MORPHOLOGIC DIAGNOSIS: Liver: Fibrosis (1pt), portal and bridging, (1pt) diffuse, severe, with marked ductular reaction, hepatocellular loss, (1pt), and hepatocellular lipidosis. (1pt).

NAME THE CONDITION: Congenital hepatic fibrosis (1pt.)

O/C - (1pt.)

Case 2. Tissue from an ox.

MICROSCOPIC DESCRIPTION: Liver: There is diffuse loss of normal hepatic architecture. There is marked degeneration (1pt.) and necrosis of centrilobular hepatocytes with shrinkage and accumulation of innumerable poorly defined cytoplasmic vacuoles (1pt.) (degeneration) and often pyknotic to rrhectic nuclei (necrosis.) (1pt.) Degenerating hepatocytes are often surrounded by variably dense strands of collagen (1pt.), which surround centrilobular veins and have replaced (1pt.) large amounts of the centrilobular and midzonal hepatocytes. Admixed within the collagen are numerous vacuolated triangular to polygonal cells which often form small aggregates (lipogranulomas) (1pt.). These bands of collagen and fibroblasts extend into the surrounding remaining periportal hepatocytes, separating and surrounding atrophic hepatocytes. (1pt.) Fibrous bands also extend into and bridge portal areas (1pt.), separating portal triads. There is marked biliary hyperplasia (1pt.). Bands of fibrous connective tissue contain low numbers of neutrophils, macrophages and lymphocytes. (1pt.) Scattered throughout the section are rare viable hepatocytes with nuclei that are 2x or more the size of surrounding hepatocytes (megalocytes). (2pt.)

MORPHOLOGIC DIAGNOSIS: Liver: Fibrosis (1pt.), portal and centrilobular, bridging, diffuse, severe, with marked hepatocellular degeneration, necrosis, and loss (1pt.), numerous intrasinusoidal macrophages (1pt.) and megalocytosis. (1pt).

CAUSE: Pyrrollizzidine alkaloid toxicosis (3pt.)

O/C - **(1pt.)**

Case 3. Tissue from a dog.

MICROSCOPIC DESCRIPTION: Liver: Diffusely, hepatic lobules are decreased in size (2pt), and hepatocyte size is also decreased (1pt) (atrophy) (1pt). (compare the hepatocytes with the size of the Kupffer cell nuclei). As a result, portal triads appear closer to each other. (1pt) Within portal areas, portal veins are absent (2pt), and there are often multiple cross-sections of tortuous thick-walled (hyperplastic) arterioles (2pt). Portal and sublobular lymphatics are dilated (1pt). There is mild replication of bile ducts within portal areas. (1pt) Small aggregates of lipid- and hemosiderin laden macrophages (lipogranulomas) (1pt) are scattered throughout the section.

MORPHOLOGIC DIAGNOSIS: Liver portal veins: Hypoplasia (1pt), diffuse, severe, with lobular (1pt) and hepatocellular atrophy (1pt).

NAME THE CONDITION: Portal vein hypoplasia (congenital portal shunt or microvascular dysplasia OK) (2pt.)

NAME TWO ASSOCIATED CLINICOPATHOLOGIC ABNORMALITIES: Elevated postprandial serum bile acids, hypoalbuminemia, hypoglobulinemia, hypoglycemia, decreased BUN, hypocholesterolemia, hyperammonemia with formation of ammonium biurate crystals in alkaline urine (2 pt)

O/C - **(1pt.)**

Case 4. Tissue from a horse.

(There are four section of liver on the slide – two are very poorly preserved due to marginal fixation, and the other two are poorly preserved at the edges. But there is definitely enough here to fully describe and make a diagnosis).

MORPHOLOGIC DESCRIPTION: Liver: There is diffuse hepatocellular degeneration (1 pt.), necrosis (1 pt.) and loss (1 pt.) affecting all parts of the hepatocyte lobule (massive necrosis) (2 pt.), but most severe in centrilobular and midzonal regions (1 pt.). Within these areas, there is loss of hepatic plate architecture (1 pt.) (resulting from loss of hepatocytes themselves), abundant hemorrhage (1 pt.), and infiltration by of moderate numbers of macrophages which have foamy cytoplasm (1 pt.) and low numbers of neutrophils and siderophages. Kupffer cells are prominent. Remaining hepatocytes, primarily in periportal areas (1 pt.) are swollen, deeply eosinophilic, and contain large clear lipid vacuoles (1 pt.). Portal areas contain multiple profiles of bile canaliculi (most likely just more prominent as a result of hepatocellular loss), and biliary epithelium is occasionally disassociated or contains one or more clear vacuoles (lipid). (1 pt.)

MORPHOLOGIC DIAGNOSIS: Liver: Degeneration (1 pt.), necrosis (1 pt.) and loss (1 pt.), massive, diffuse, severe, with stromal collapse and hepatocellular lipidosis. (1 pt.)

NAME THE CONDITION: Theiler's disease (serum hepatitis OK) (2pt.)

CAUSE: Equine flavivirus or equine parvovirus (at the moment) (1 pt.)

O/C: **(1 pt.)**